

hydrate metabolism than has been previously recognized, it cannot be maintained that we have as yet succeeded in obtaining unequivocal evidence that the phenomena described are due to the excessive influence of the adrenal internal secretion or to the exclusion of this secretion from the circulation. Some of these phenomena can probably be equally well accounted for on the supposition that imperfectly understood nervous factors have been brought into play. It must, therefore, be left to further experiments, in which close attention is given to control observations on the nervous system, to determine whether the internal secretion hypothesis is tenable.

A REPORT OF TWO CASES OF MULTIPLE SCLEROSIS, WITH NECROPSY:

WITH REMARKS ON MUSCULAR ATROPHY, SECONDARY DEGENERATION, AND LOSS OF TENDON REFLEXES, WITH INCREASED MUSCULAR TONICITY, OCCURRING IN THIS DISEASE.¹

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MULTIPLE sclerosis seems to be a rare disease in America, and scarcely any cases with necropsy are to be found in the literature of this country. I am inclined to think that some cases are overlooked, and that the disease, while uncommon, does occur more frequently than the paucity of records gives us reasons to believe. B. Sachs remarked at the time he wrote his critical digest on multiple sclerosis, in 1898, that he had been able to find only one necropsy record of a case of this disease published in America, and that was by Seguin, and was incomplete.

Since the publication of Sachs' paper a case with necropsy has been reported by Burr and McCarthy² (1900), and another that probably should be classed under multiple sclerosis, although published only in abstract, May, 1902, by J. R. Hunt.³ The paper by E. W. Taylor⁴ is one of the best on the subject by American writers, but the cases he reports occurred in Germany, and are a contribution to German literature.

I report two cases, with microscopic examination of the nervous tissues. The first case was in the service of Dr. F. X. Dercum, at the

¹ Read in abstract at the annual meeting of the American Neurological Association, June 1902.

² Sachs. *Journal of Nervous and Mental Disease*, 1898.

³ Burr and McCarthy. *Ibid.*, 1900.

⁴ J. R. Hunt. *Ibid.*, May 12, 1902, p. 288.

⁵ E. W. Taylor. *Deutsche Zeitschrift für Nervenheilkunde*, vol. v.

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Philadelphia Hospital, and is a typical example of the disease. I am indebted to Dr. Dercum for the clinical history and the pathological material. The second case was in my service at the Philadelphia Hospital, and forms a striking contrast in its clinical aspects to Dr. Dercum's case.

CASE I.—C. O., laborer, white, aged twenty-five years, was admitted to the Philadelphia Hospital, March 26, 1900. A brief history was taken at this time.

November 6, 1900. Chief complaint: stiffness and weakness of right upper and lower limbs, vertigo, and tremor.

Father died, at the age of fifty years, from gastric disease; mother died after labor; one brother died in infancy. Three sisters and four brothers are living and well.

The patient had measles when a child and typhoid fever seven years ago. He has used alcohol and tobacco to excess, but denies venereal disease.

Two years ago last winter he worked four or five hours one night in the hold of a vessel with nothing on but trousers, drawers, shoes, and stockings. Thus clad he came up on the deck, when it was bitterly cold and raining sleet and hail, and took a drink of ice-water. He remained on deck about twenty minutes, not noticing the cold, and then returned to the hold, where he worked for six hours more. In the morning he went home and to bed, still experiencing no ill effects from the exposure. That afternoon he felt stiff all over, and the stiffness remained for several days. Four days after this exposure he had to work seven hours in the hold of a ship shovelling wet coal. While shovelling he stood in the wet coal, and afterward had to sit in the wet, shivering, while waiting for more coal. The next morning he went directly from the hold to the deck, to sweep. As soon as he reached the deck he began to feel stiff, and staggered and was hardly able to walk. That night he experienced sharp pains in the thighs, more in the right thigh, and the stiffness was most marked on the right side.

This condition remained stationary for one month, when he awakened one morning with sharp pains in the right leg, with marked rigidity of that member. Soon after this he suffered from similar pains in the left leg, although not so severely.

His condition improved slightly for several months following, but there has been little change during the past year. He has occasionally had loss of sphincter control during the past year.

Present Condition (November 6, 1900). The patient is a well-developed young man. His intelligence is not impaired, but his speech is slow and scanning. Pupils are equal and the irides respond promptly to light and in accommodation. The ocular muscles are apparently normal. There is a slight tremor of the tongue. The tendon reflexes of the upper limbs are normal (later those of the right upper limb were exaggerated). The epigastric reflex is normal, but the abdominal and cremasteric reflexes are absent. The patellar reflex is exaggerated on each side, more so on the right side. Ankle clonus is present on each side. Babinski's reflex is present. Grip of the right hand by dynamometer is 41; of left, 45. He has apparently very little loss of power in the upper or lower limbs. Temperature sense is normal,

except in the feet, where there is delayed sensation, and in the toes and heel of the right foot, where there is reversal of temperature sense. Tactile and pain senses are unimpaired. Station is poor. He sways with his eyes open and falls when they are closed. Gait is staggering and the right foot is dragged. Some ataxia is observed in the upper limbs, especially on the right side. Intention tremor is present in the right upper limb.

The report of the examination of the eyes by Dr. G. E. de Schweinitz, November 18, 1900, is as follows: "Light reflex normal. Each disk atrophic; atrophy more marked on temporal side. No change in vessels except a slight perivasculitis. Well-marked nystagmus."

December 8, 1901. He has wasted very rapidly during the past two months, and during the past month has had almost absolute loss of power in his lower limbs, especially on the right side. The wasting is general. Breath smells sweetish, and examination of urine shows sugar in large amount.

The patient died December 9, 1901.

The microscopic examination of the brain and spinal cord, made by me, revealed numerous areas of sclerosis, especially in the cord. It is not necessary to dwell on the pathological condition, as it was like that commonly seen in multiple sclerosis. The case was clinically and pathologically a typical one of this disease. I had the opportunity to examine this patient frequently during his residence in the hospital.

The causative relation of cold and dampness to multiple sclerosis seems to be clearly shown in this case. Krafft-Ebing has insisted on this etiology of the disease, but it is seldom that the symptoms follow so closely the exposure, as in this case. The man C. O. developed the symptoms of multiple sclerosis after repeated and long exposure to cold and dampness in shovelling coal in the hold of a vessel. Residence in a damp house was believed to be the cause of the disease in the second case. It is probable that this was not the only cause in either case.

CASE II.—The patient, a woman aged fifty-one years, was in my service at the Philadelphia Hospital during the summer of 1901. She said the first symptoms of her disease began in 1891 or 1892 after a residence in a damp house. She denied venereal disease and abuse of alcohol, although she acknowledged that she had used much tobacco. Impaired gait was noticed early, but the gait soon improved, and later became worse again. She was able to walk until 1899, but staggered so that she had to be supported. In 1891, while doing housework, she suddenly became numb in the feet, and the numbness extended gradually up the limbs, with progressive loss of power. In two days the numbness had extended to the waist, and she had a girdle sensation of pressure and incontinence of urine and feces. Her speech was slow, and she had some difficulty in saying words. My notes, made June 24, 1901, are as follows:

The lower limbs appear to be completely paralyzed, and although they are drawn up at times, the movement is probably entirely involuntary. When told to move her toes she is unable to do so. The lower limbs are wasted. The right thigh in its middle portion measures

thirteen inches; the left thigh in the corresponding portion measures fourteen inches. The thighs are proportionately more atrophied than the legs below the knees, but the atrophy in the latter is partly masked by œdema. The feet are very œdematous and pit on pressure. Pressure over either leg causes the leg to be drawn up; this is especially true of the left lower limb. The movement is the result entirely of reflex action, and is involuntary. The knees and feet are inverted, and there is much spasticity of the lower limbs, which is greatly increased by passive movement. *There is no knee-jerk on either side, even with reinforcement.* This is especially noteworthy because of the spasticity in the lower limbs. The knee-jerk is not prevented by the spasticity, as the spasticity is comparatively slight when the limbs have not been irritated, and even without irritation of the limbs there is no knee-jerk. When the attempt is made to obtain the knee-jerk there is no contraction visible in the quadriceps muscle of either side. Ankle clonus is not obtained on either side. The Babinski reflex is very pronounced on each side, the great toe as well as the other toes being drawn forcibly upward. The Achilles jerk is absent on each side. Sensation for touch, pain, and temperature is normal in the lower limbs. No tenderness is produced by pressure of the nerves or muscles.

The movements in the upper limbs are free, and the resistance to passive movements in these limbs is proportional to the muscular development. The upper limbs above the elbow are fairly well developed. The interossei muscles, the thenar and hypothenar eminences are much wasted, and the grasp of each hand is almost *nil*. The forearms are wasted, but not intensely so. Sensation for touch, pain, and temperature is normal in the upper extremities and over the chest. The tendon reflexes of the upper limbs, biceps tendon, triceps tendon, and wrist-jerks on either side are not strikingly abnormal. No tenderness is felt on pressure of nerves or muscles.

The cranial nerves are not distinctly implicated. The tongue is protruded straight. The masseters contract firmly. The corner of the mouth is drawn up well on each side, and the eyes are firmly closed. At the present examination there is considerable swelling of the left eyelid.

The spinal column is normal.

The mentality is feeble. The patient replies correctly to questions, but her statements concerning her past condition are not fully reliable. She complains of numbness in her hands. She has incontinence of urine and feces.

The report of the examination of her eyes, made by Dr. H. F. Hansell, June 24, 1901, is as follows:

"Paralysis of the third nerve of the right side and also of the left, amounting to almost complete ophthalmoplegia. Pupillary response is good. Pupils are equal. Partial atrophic cupping of both optic nerves, with reduction in size of the bloodvessels, without signs of previous inflammation."

The patient grew gradually worse, and died July 5, 1901.

The microscopic examination, made by me, gives the following results:

Numerous sclerotic areas are found throughout the brain and spinal cord. One such area is found implicating the nuclei of the oculomotor, trochlear, and abducent nerves, but is not confined to these nuclei. The nerve cell-bodies of the oculomotor nuclei are not very distinctly diseased, as shown by the thionin stain, and yet some may be slightly altered. The posterior longitudinal bundle on each side is in the sclerotic area, and contains at this part a few medullated nerve fibres. Half of the medulla oblongata at one level has undergone a sclerotic change. A sclerotic area is found in the left optic nerve, but the right optic nerve appears to be normal.

Some degeneration, as shown by the Marchi method, is detected in the sclerotic areas of the spinal cord. Secondary degeneration of moderate intensity is found in each crossed pyramidal tract, below the

FIG. 1.



CASE II.—Section through the nuclei of the oculomotor nerves, showing a sclerotic patch implicating these nuclei.

mid-thoracic region, but is greater on one side. No sclerotic areas are found in the posterior columns at the upper lumbar region, but in the mid-lumbar region a sclerotic area is present on each side of the posterior septum, and implicates the posterior root fibres after their entrance into the posterior columns. These areas do not extend into the lower lumbar region, but here a sclerotic area is found implicating one anterior horn and the antero-lateral column. A sclerotic area implicates the greater part of the left anterior and lateral columns and the gray matter of the left side and a part of the left posterior columns in the sacral region. A sclerotic area is found also in the right lateral column, and gray matter of the right side in the sacral region. Many of the nerve cell-bodies of the anterior horns of the cervical and lumbar region appear shrunken and have imperfect dendritic processes, and some are much pigmented. The thionin stain was employed. The

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nerve cell-bodies in a sclerotic area implicating one anterior horn in the lower lumbar region resemble very closely those of the other horn

FIG. 2.



CASE II.—A sclerotic area implicates a large portion of the medulla oblongata.

FIG. 3.



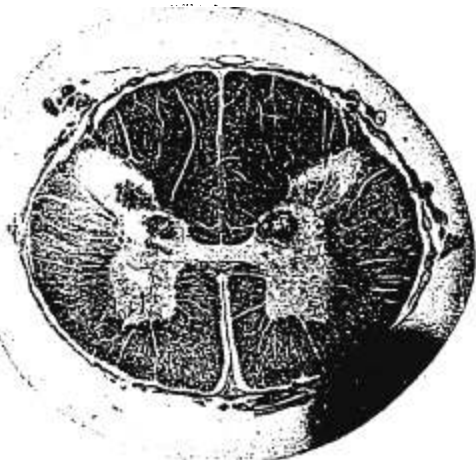
CASE II.—Section from the cervical region, showing a large sclerotic patch on each side of the spinal cord.

in the same section where there is no sclerotic area, and in neither horn are they normal. A branch of one of the plantar nerves is partially degenerated. Many other sclerotic areas not described are found in the brain and spinal cord.

A summary of the case is as follows:

In 1891 the patient—a woman, then about forty-one years of age—complained of numbness in the feet, and soon the legs became numb. She had at this time progressive loss of power in the lower limbs and

FIG. 4.

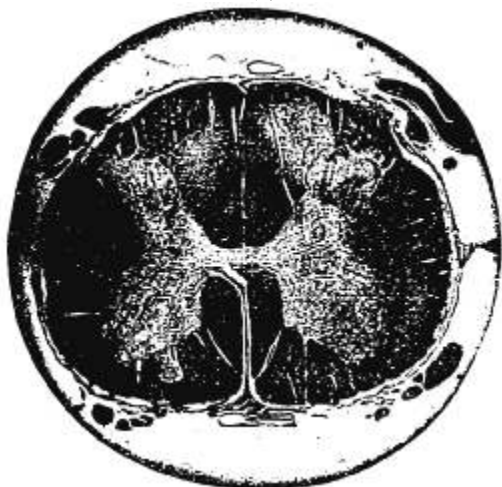


CASE II.—No sclerotic area is seen in the posterior columns in this section from the upper lumbar region. The secondary degeneration of the crossed pyramidal tracts is not well reproduced in the photographs.

staggering gait. Within two days the numbness extended as high as the waist, and she complained of girdle sensation, and had complete paralysis of the lower limbs. She had also incontinence of urine and feces. She had resided in a damp house. The disturbance of gait soon disappeared for a time, and she was able to walk with difficulty until 1899, but staggered. She complained of shooting pains at times in the knees, extending to the ankles. In 1901 her speech was somewhat drawing. The lower limbs were completely paralyzed, were much wasted, and spastic; slight irritation causing the limbs to be drawn up involuntarily, and yet the patellar and Achilles reflexes

were absent. The knees and feet were inverted. The Babinski reflex was very pronounced. Sensation was normal everywhere. Voluntary movement in the upper limbs was impaired, and the grasp of the hands was very feeble. The hands were very much atrophied. The tendon reflexes of the upper limbs were normal. Mentality was feeble. She had almost complete external ophthalmoplegia and partial atrophic cupping of both optic nerves. Iritic response was good. The case was one of disseminated sclerosis, as shown by the microscopic examination.

FIG. 5.



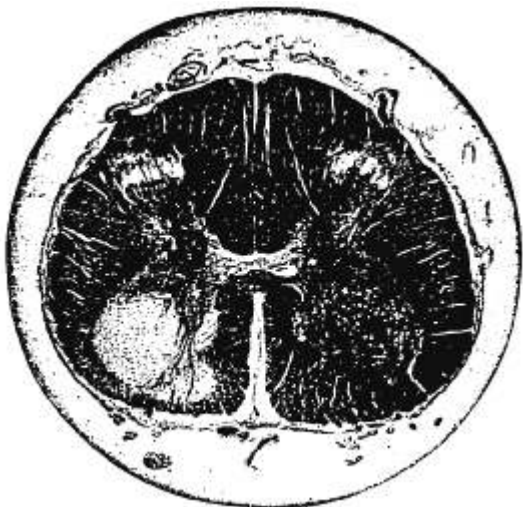
CASE II.—Section from the mid-lumbar region. A sclerotic area is seen on each side of the posterior septum only in the mid-lumbar region (compare Fig. 5 with Figs. 4 and 6). The tendency to symmetry of the areas in multiple sclerosis is well shown in this section.

The interesting features of this case were: The pronounced muscular atrophy; the secondary degeneration of the crossed, pyramidal tracts, more distinct, however, on one side, in a case of disseminated sclerosis; the loss of the patellar and Achilles reflexes, with spasticity of the lower limbs, the loss of these reflexes probably being the result of sclerotic areas in the posterior columns and a part of the anterior horns of the lumbar and sacral regions; and the almost complete external ophthalmoplegia resulting from a sclerotic area in the nuclei of the third, fourth, and sixth nerves.

The muscular atrophy was very pronounced in the lower and upper limbs, and in the latter the atrophy was more distinct in the small muscles of the hands. The atrophy in the lower limbs below the knees was partly masked by oedema. Oppenheim¹ regards muscular atrophy as a rare sign of multiple sclerosis, and states that, as a rule, the muscles preserve their normal size and electrical reactions.

L. Brauer² believes that muscular atrophy is very uncommon in the early stages of multiple sclerosis, and he reports a case of this disease

FIG. 6.



CASE II.—Section from the lower lumbar region. The sclerotic areas of the posterior columns represented in Fig. 5 have disappeared, but a sclerotic area is seen in one anterior horn extending into the white matter.

in which muscular atrophy was an early symptom and later became very pronounced. He acknowledges that muscular atrophy does occur occasionally in multiple sclerosis, and he believes it affects especially the small muscles of the hand and the peroneus distribution. He refers to cases of muscular atrophy occurring in multiple sclerosis reported

¹ Oppenheim. *Lehrbuch der Nervenkrankheiten*, third edition, p. 314.

² Brauer. *Neurologisches Centralblatt*, July 15, 1898, No. 14, p. 635.

by Jolly, Ebstein, Leube, Schüle, Buchwald, Otto, Engesser, Dejerine, and Pitres. Killian also is said to have observed muscular atrophy in multiple sclerosis.

Probst¹ speaks of the infrequency of muscular atrophy in multiple sclerosis, and refers to a few cases in which it was observed. In most of these cases no very important alteration of the cell-bodies of the anterior horns was seen, but Probst was able to find a decrease in the number and a shrinkage of the cell-bodies in the anterior horns of the upper cervical region in one case of multiple sclerosis with muscular atrophy, although no sclerotic focus could be found in this region. This case Probst pronounces as unquestionably one of multiple sclerosis. Alteration of the nerve cell-bodies was observed by me in my case. According to Probst, alteration of nerve cell-bodies in multiple sclerosis has been observed by Bourneville, Guérard, Charcot, Frommann, Gowers, Obersteiner, Frommann, Taylor, Schuster, and Bielschowsky; but muscular atrophy does not appear to have been observed in all these cases in which cellular change existed.

Secondary degeneration is not seen in multiple sclerosis, according to some writers, and yet in my case it could be found in the crossed pyramidal tracts in the spinal cord below the mid-thoracic region. According to Probst,² Schultze was the first to offer as an explanation of the absence of secondary degeneration the preservation of the axons in the sclerotic foci. Secondary degeneration in multiple sclerosis is said to have been observed by Jolly in 1872, Buss in 1889, Werdnig in 1888, Babinski in 1885, Redlich in 1895, and Rossolimo—certainly a small number of cases, in consideration of the large number of cases of this disease published. In most of the cases in which secondary degeneration was found it extended only a short distance, and was only of moderate degree. In a case reported by Probst³ secondary degeneration of the anterior and lateral pyramidal tracts existed, and extended from the proximal portion of the pons into the lumbar region. Probst mentions that possibly some might regard his case as one in which multiple sclerosis was combined with amyotrophic lateral sclerosis, inasmuch as the nerve cell-bodies of the anterior horns were not normal and sclerotic foci were not found in the spinal cord. This combination, I think, might occur, as both diseases possibly are the result of faulty development of nervous tissue in the embryo.

Since my paper was read before the American Neurological Association an interesting article in which the occurrence of secondary degeneration in multiple sclerosis is considered has been published by Schupfer.⁴ He says secondary degeneration in this disease has been observed only

¹ Probst. *Deutsche Zeitschrift für Nervenheilkunde*, 1896, vol. xii.

² Loc. cit.

³ Loc. cit.

⁴ Schupfer. *Monatsschrift für Psychiatrie und Neurologie*, August, 1902, p. 109.

by Jolly, Westphal, Schultze, Greiff, Babinski, Werdnig, Buss, Rosso-limo, Probst, Thomas and Long, and Taylor. He subjects these cases to a critical study, and concludes that only in the cases of Buss, Probst, and Thomas and Long was the degeneration of the pyramidal tracts in connection with lesions in the spinal cord, medulla oblongata, and pons. In Thomas and Long's case the cause of the degeneration he thinks was syphilitic endarteritis. Degeneration of the pyramidal tracts is therefore, in his opinion, extremely rare in multiple sclerosis, and may be caused by sclerotic foci or may occur independently of these. The secondary degeneration of the pyramidal tracts may be associated with diffuse sclerosis of the cerebral hemispheres with atrophy of the gyri and alteration of the cells of the central gyri, as in the cases of Schultze and Greiff. The occurrence of degeneration of the pyramidal tracts without focal lesions he finds difficult to explain. It is possible that Schupfer does not lay sufficient importance upon the short extent of some of the foci in multiple sclerosis, and important areas of sclerosis of limited extent may be overlooked. In Jolly's case and Schultze's, Schupfer says the sclerotic areas do not explain the degeneration of the pyramidal tracts, as the degeneration began at a part where sclerotic areas were not detected. It is important to bear in mind the cumulative effect of slight degeneration at different levels; this, however, Schupfer does not overlook.

The loss of the patellar reflexes, with increase in the muscular tonicity of the lower limbs, was noteworthy. There could be no mistake in regard to this, because in my notes made at the time of the clinical examination I underscored twice the statement bearing on this point. The statement reads: "The knees and feet are inverted, and there is much spasticity of the lower limbs, which is greatly increased by passive movement. *There is no knee-jerk on either side, even with reinforcement.* This is especially noteworthy because of the spasticity in the lower limbs. The knee-jerk is not prevented by the spasticity, as the spasticity is comparatively slight when the limbs have not been irritated, and even without irritation of the limbs there is no knee-jerk. When the attempt is made to obtain the knee-jerk there is no contraction visible in the quadriceps muscle of either side. Ankle clonus is not obtained on either side. . . . Achilles jerk is absent on each side."

Marie¹ has said that the tendon reflexes are never absent in multiple sclerosis.

The difference in the condition of muscular tonus and of the tendon reflexes in my case is most extraordinary. Van Gehuchten² has paid much attention to the relation of the muscular tonus to the tendon

¹ Marie. *Leçons sur les maladies de la moëlle.*

² Van Gehuchten. *Journal de Neurologie*, August 20, 1897, No. 16, p. 305.

reflex. He believes there is a certain parallelism between the two; but the parallelism does not always exist, and the tendon reflexes may be exaggerated when the muscular tonus is not exaggerated, or the tendon reflexes may be preserved when muscular atony is found, or they may be absent when muscular hypertonia or contracture exists. I shall not discuss the theories he advances to explain these conditions, as they do not seem to me to be sufficiently founded on fact.

Strümpell¹ also believes that exaggeration of the tendon reflexes is often, but not always, associated with hypertonia of the muscles.

On the other hand, Parhon and Goldstein² do not believe that the condition of the reflexes is independent of the condition of the muscular tonus, and they assert that hypertonicity never occurs when the reflexes are lost, unless the reflexes are lost because the rigidity is so great that no movement is possible. These views are held also by some American authors. I show by this case that the patellar reflex may be lost when hypertonicity of the lower limbs is present and when this abolition of the reflex is not the result of immobility of the limbs from excessive rigidity. I have not been able to obtain Parhon and Goldstein's original paper, and must depend upon an abstract.

It is possible that in my case the patellar and Achilles reflexes were lost because sclerotic areas existed in the regions in which these reflex arcs within the spinal cord were contained. In the upper lumbar region no sclerotic focus was found, but in the mid-lumbar region a sclerotic patch was found in each posterior column implicating the fibres of the posterior roots after they had entered the cord. These sclerotic patches disappeared in the lower lumbar region, but at this level a sclerotic patch was found on one side implicating the anterior horn and anterior roots within the cord. The sclerotic areas found in the sacral cord were probably the cause of the lost Achilles jerk, and, in addition, the degeneration of one of the popliteal nerves examined may indicate that some of the terminal portions of the peripheral nerves were diseased. This alteration of the nerve fibres of the peripheral nerve was probably secondary to the alteration of the cell-bodies of the anterior horns of the spinal cord. The loss of the tendon reflexes of the lower limbs seems easily explained.

The exaggeration of the muscular tonicity may have been caused by the implication of the pyramidal tracts in the sclerotic areas above the lumbar region. If the impulses from all parts of the lower limbs had been almost entirely cut off it seems hardly probable that the muscular tonicity would have been exaggerated. A slight interference with the sensory impulses concerned in the reflexes may, however, be sufficient

¹ Strümpell. *Neurologisches Centralblatt*, July 1, 1899, No. 13, p. 618.

² Parhon and Goldstein. *Roumanie médicale*, 1899. Abstract in *Revue Neurologique*, March 15, 1902, p. 223.

to cause a loss of these reflexes, even in association with hypertonicity. Doubtless sufficient sensory impulses passed to the cord to cause exaggeration of the tonicities when the pyramidal tracts were diseased and the inhibition from the brain was removed.

In combined systemic disease the posterior and lateral columns are degenerated, and in some cases the spasticity later yields to flaccidity, apparently because after a certain time sensory impulses from the limbs to the cord are almost cut off, and therefore the paralysis becomes flaccid, even though the lateral columns may continue to degenerate. In illustration I refer to a case reported by Rheinbaldt,¹ in which rigidity of the lower limbs was prominent in the commencement of the palsy of these limbs, but when the patient came under Rheinbaldt's observation the palsy had become flaccid. Systemic degeneration of the direct and crossed pyramidal tracts, of the direct cerebellar tracts, and of the posterior columns was found. Rheinbaldt refers to a still more satisfactory case reported by Rothmann,² in which rigidity of the lower limbs gave place within two months to flaccidity.

Raymond and Cestan³ have reported a very interesting case of disseminated sclerosis with paralysis in the associated movements of the eyeballs. The eyeballs were in a median position, and there was no ptosis. The irides reacted well to light and in accommodation. In each lateral movement of the eyeballs toward the right or toward the left the cornea was arrested about 7 or 8 millimetres from the external angle. Convergence, however, was very slightly affected. A sclerotic plaque was found implicating the nuclei of the third and fourth nerves. They quote Parinaud as saying that these associated paralyses are common in multiple sclerosis, but are usually incomplete. The ocular paralysis in Raymond and Cestan's case was incomplete, as it was in mine, and the lesions in the ocular nuclei in the two cases were similar. In a note of June 24, 1901, by Dr. H. F. Hansell, regarding my case, the statement is made that there was paralysis of the third nerve of each side and almost complete ophthalmoplegia. Iritic response was good. The pupils were equal. There was partial atrophic cupping of both optic nerves, with reduction in size of the bloodvessels without signs of previous inflammation. Dr. Hansell, in answer to an inquiry made by me, says: "'Almost total ophthalmoplegia' means in this case that the sixth nerves were also involved. Had external rotation been preserved my expression would have been 'double third-nerve paralysis,' and the word 'ophthalmoplegia' would not have been mentioned. Again, the total third-nerve paralysis could not have been present, since the report states that the pupils were responsive to light."

¹ Rheinbaldt. *Archiv für Psychiatric*, vol. xxxv., No. 1, p. 57.

² Rothmann. *Deutsche Zeitschrift für Nervenheilkunde*, vol. vii.

³ Raymond and Cestan. *Revue Neurologique*, January 15, 1902, p. 52.

The implication of the sixth nerves in my case, as in Raymond and Cestan's, suggested a paralysis of associated movement resulting from the destruction of the connection by means of the posterior longitudinal bundle between the nuclei of the oculomotor nerves and those of the abducent nerves. This form of paralysis is known by the Germans as *blicklähmung*. In my case at least the sixth nuclei as well as the third and fourth were implicated in the sclerotic area.

EOSINOPHILIA IN PELVIC LESIONS AND IN THE VERMIFORM APPENDIX.

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In the routine microscopic examination of all tissues removed at operations in the gynecological service of the Lakeside Hospital, cells with eosinophilic granulations have frequently been observed in the tissues, especially in inflammatory conditions. In this laboratory their occurrence was first noted in the gynecological department in 1898, and subsequently so many instances were observed in all the departments of the laboratory that Howard and Perkins¹ were able to report a large number of cases. The present article deals with the material of the gynecological department alone.

Howard and Perkins in material from 825 consecutive operations in the different services of the hospital found eosinophiles in the tissues of 108 cases, or 13 per cent. Of these 80, or 74 per cent., were inflammatory; 24 cases were acute and 56 were chronic; 9 were tuberculous and 9 were syphilitic. In 120 consecutive autopsies eosinophiles in larger or smaller numbers were found in organs showing pathological changes in 27 instances. They occurred in the spleen, liver, stomach, intestines, kidney, heart, lung, thymus, lymph glands, and skeletal muscles.

These findings appear to warrant the conclusion that eosinophiles take an active part in many inflammatory processes, more especially appendicitis, pyosalpinx, and ovarian abscess, as well as in many cases of carcinoma showing inflammation; that they are often associated with polymorphonuclear neutrophils and almost invariably with plasma cells; that their development from plasma cells and mononuclear leucocytes could be traced in many instances. These writers are of the opinion that the eosinophiles found in the tissues have usually

¹ Johns Hopkins Hospital Reports, vol. x.